# Risk Factors and Management Approach for Deep Sternal Wound Infection After Cardiac Surgery at a Tertiary Medical Centre

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*Background:* Deep sternal wound infection (DSWI) is a rare but severe complication following cardiac surgery. Our study investigated the risk factors and treatment options for patients who developed DSWI at our institution between May 1988 and April 2008.

*Method:* Data was collected prospectively in a database and information on demographics reviewed retrospectively on 5649 patients who underwent cardiac surgery during this period.

Results: The incidence of DSWI was 34/5649 (0.6%). These patients were older (mean age 66.1 vs. 64.5), more likely to die (in hospital mortality 11.8% vs. non DSWI group 1.8%) and had longer hospital stays (DSWI group mean stay 25 days vs. non DSWI group 9 days). Using Fisher's exact test the risk predictors for DSWI determined at our institution included diabetes managed with oral medications (p = 0.021), previous cardiac surgery (p = 0.038), BMI  $\geq 30$  (p = 0.041), LVEF  $\leq 30$  (p = 0.010), IABP usage (p = 0.028) and homologous blood usage (p < 0.001). Most commonly bilateral pectoralis major muscle flap (BPMMF) was used for treatment of DSWI (11/30, 36.7%).

Conclusion: Ultimately our data was comparable to published data in the literature on known risk predictors.

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# Introduction

Post sternotomy deep sternal wound infection (DSWI) also commonly known as mediastinitis is a severe complication of cardiac surgery which contributes significantly to patient morbidity and mortality and overall cost to the healthcare system [1–4]. The frequency of this complication varies globally partly due to definitions used, however some centres have reported rates between 0.2% and 8% [5] and others ranging between 0.25% and 4% [6] of cardiac surgeries. In Australia, approximately 23,000 cardiac surgical procedures are conducted annually [7]. A study by Robinson et al. assessed the risk factors associated with DSWI in Victoria and showed an incidence of 1.3% of near 12,000 cardiac patients operated on. There has yet to be any other state based assessment in Australia [7].

Of greater consequence however are the reported mortality rates which vary from 8% to 45% [4,6]. Furthermore DSWI is associated with significant morbidity and prolonged hospital stays. Collectively this equates to significant cost to the healthcare system. One study conducted in New Zealand by Upton et al., showed an almost doubling in the cost per patient who underwent cardiac surgery and developed post operative *Staphylococcus aureus* mediastinitis compared with those patients who followed an uncomplicated post operative course [8]. The cost was attributed to the increased mean length of stay and subsequent operative intervention was required.

The pathogenesis of DSWI involves a complex multifactorial process and includes a number of varying microbiological organisms. These range from Grampositive and Gram-negative organisms and also include fungi. The most common pathogens reported however continue to be *Staphylococcus epidermidis* and *Staphylococcus aureus* which comprise part of the normal commensal flora of the skin [9,10]. *Staphylococcus epidermidis* has been reported in recent times to be responsible for 43–64% of all DSWIs and of this number as many as 75% have been

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found to be methicillin resistant. Clinically it appears that *S. epidermidis* follows a more insidious course and often patients reveal very little in regards to symptomatology. *Staphylococcus aureus* in contrast tends to behave more aggressively and greater clinical evidence of infection is apparent [9].

Several risk factors both patient and procedural dependent have been identified for DSWI. Several European studies and more recently a state based study by Robinson et al. in Victoria, Australia, have focused on these. Preoperative or patient factors include diabetes, obesity, smoking, COPD, low LVEF and renal failure to name a few. Intraoperative factors such as length of surgery and the use of bilateral internal mammary artery conduits have been considered important to the development of DSWI. Furthermore prolonged ventilation, redo-operation, requirement of transfusion and prolonged use of inotropic drugs as post operative factors in some studies have also shown association with DSWI [1–3,6,7,9,10].

Ultimately although extensively investigated and reported in Europe and overseas, little has been published in Australian centres regarding incidence, risk factors and management approach of DSWI. Our study describes the risk factors, management approach and follow-up functional status in patients who had DSWI at a tertiary medical centre over a 10 year period.

## Materials and Methods

# Patient Selection

The study was conducted at the Department of Cardiothoracic Surgery at the Princess Alexandra Hospital in Brisbane, Australia. Data was collected prospectively on all cardiac surgical patients operated between May 1998 and April 2008 in a systemised database. This database was retrospectively reviewed and the data of 5649 patients was analysed for the study. Patient charts were retrieved when necessary for completion of the data. Deep sternal wound infection incidence of 34/5649 (0.6%) was recorded for the period and data on management, outcome and followup of these patients with DSWI was retrieved. Patients who underwent sterile dehiscence of superficial sternal wound infections were not included. All surviving patients were followed up either in the outpatient clinic or by telephone interview. Table 1 shows the comparison of patient demographics and operative variables of those who did not develop DSWI and in those who did.

### Surgical Management

Management of patients who developed DSWI was determined by surgeon preference. It varied from conservative management with antibiotic therapy and dressing, to surgical debridement and rewiring to muscle flap closure. Muscle flap in our institution was performed by plastic and reconstructive surgeons. Flap closure was used as a first line after debridement or after failed wiring. Muscle flap coverage occurs as a two step procedure. Stage 1: debridement with saline packs every four hours associated with

Table 1. Patient Demographics Stratified by DSWI.

	0 1	, ,		
Demographic	No	DSWI	p Value	
0 1	DSWI	n = 34	•	
	n = 5615	(0.6)		
	(99.4)			
Preoperative				
Age, years	64.5/65.8	66.1/64.1	_	
(mean/median)				
Smoker				
Previous	3113	23 (67.6)	0.051	
	(55.4)	, ,		
Current	453 (8.1)	4 (11.8)	0.159	
Diabetes				
Oral	884	10 (29.4)	0.021	
medica-	(15.7)			
tion				
Insulin	246 (4.4)	1 (2.9)	0.346	
Steroids	85 (1.5)	0 (0)	0.596	
Previous	230 (4.1)	4 (11.8)	0.038	
cardiac				
surgery				
BMI ( $kg m^{-2}$ )				
≥30	1760	15 (44.1)	0.041	
	(31.3)			
$\geq$ 40	90 (1.6)	0 (0)	0.578	
$LVEF \le 30$	433 (7.7)	7 (20.6)	0.010	
Operative status				
Elective	3910	18 (52.9)	0.017	
	(69.6)			
Urgent	1503	13 (38.2)	0.049	
	(26.8)			
Operation		()		
CABG	3686	23 (67.6)	0.141	
	(65.6)	- (0.0)		
AVR	594	3 (8.8)	0.222	
	(10.6)	2 (0.0)	0.400	
1 (T/D /D )	338 (6.0)	3 (8.8)	0.192	
MVR/Repair	260 (6.6)	4 (11 0)	0.114	
CARC AND	369 (6.6)	4 (11.8)	0.114	
CABG + AVR	EC (1.0)	1 (2.0)	0.045	
Aortic	56 (1.0)	1 (3.0)	0.247	
dissection Perioperative				
Perioperative	88/80	99/93		
Bypass time min	00/00	77173	_	
time, min (mean/median)				
(mean/median) IABP	115 (2.0)	2 (8 8)	0.028	
Homolo-	115 (2.0)	3 (8.8)	< 0.028	
gous blood	(19.2)	21 (61.8)	<0.001	
O	(17.4)			
usage				

Values are expressed as number of patients (percentage of either DSWI group or no DSWI group) or as a mean/median or minutes. AVR, aortic valve replacement; BMI, body mass index; CABG, coronary artery bypass graft; Diabetes, Patients preoperatively were either treated with oral medications or insulin; IABP, intraaortic balloon pump; MVR/Repair, mitral valve replacement/repair; LVEF, left ventricular ejection fraction (expressed as percentage).

intravenous antibiotics. Flap procedure is planned when patient is afebrile for 48 hours, clean wounds, no inotrope and stable haemodynamics. Muscle flaps are raised keeping subcutaneous tissue attached. Sternum is closed with multiple 0 PDS pulley sutures through outer table only. 2× 19f Blake drains are inserted below the flaps. 0 PDS interrupted sutures are used to approximate muscles over the sternum. Rectus sheath is closed to prevent epigastric hernia. Five millilitres of tisseal is inserted below each flap. 3-0 monocryl/nylon is used for subcutaneous tissue

**Table 2.** Classification of DSWI According to El Oakley and Wright [12].

Туре	Description				
I	DSWI presenting within 2 weeks after operation in the absence of risk factors				
II	DSWI presenting at 2–6 weeks after operation in the absence of risk factors				
IIIA	DSWI type I in the presence of one or more risk factors				
IIIB	DSWI type II in the presence of one or more risk factors				
IVA	DSWI type I, II or III after one failed therapeutic trial				
IVB	DSWI type I, II or III after more than one failed therapeutic trial				
V	DSWI presenting for the first time more than 6 weeks after operation				

closure. Patients are usually brought to ICU ventilated for the first 24 hours. Drains are kept in till less than 20 ml is produced in 24 hours. Intravenous antibiotics are given for six weeks. Dressings are microfoam till drains come out and then tubigrip vest/binder after that for six weeks. No arm abduction is allowed for two weeks.

## Definitions of Mediastinitis

For the purposes of this study the definition of DSWI or mediastinitis used was provided from the guidelines of the Centre for Disease Control and Prevention 1999 [11]. According to these guidelines, diagnosis of DSWI requires at least one of the following

- (a) an organism positively cultured from the mediastinal space (tissue/fluid);
- (b) evidence of mediastinitis seen during operation;
- (c) one of the following conditions: chest pain, sternal instability, or fever (>38 °C), in combination with either purulent discharge from the mediastinum or an organism isolated from blood culture or culture of mediastinal drainage.

The patients who developed DSWI were classified according to the criteria proposed by El Oakley and Wright (Table 2) [12] which are broadly used. This classification system differentiates patients with DSWI based on time of presentation and preoperative established risk factors. The most prevalent classification was type IV with 15/30 (50%) of patients with DSWI in our study.

#### Statistical Analyses

Comparison of the categorical variables was made using Fisher's exact test of statistical significance with a p < 0.05.

#### **Results**

During a 10-year period (May 1998–April 2008) 5649 patients underwent cardiac surgery in our institution. The incidence of DSWI experienced at our institution was 34/5649 (0.6%) and of these the mean age of patients was 66.1 compared to the remaining 5615 (99.4%) patients who did not develop DSWI who had a mean age of 64.5. The

comparisons of patient demographics and intraoperative variables of these two groups are given in Table 1.

The organisms most commonly isolated in patients with DSWI were *S. aureus* in 8/34 (23.5%), MRSA in 4/34 (11.8%), *S. epidermidis* in 6/34 (17.6) and Gram negative in 5/34 (14.7). The remainder returned negative cultures.

Of the patients who were diagnosed with mediastinitis, 31 (0.9%) patients were diagnosed prior to discharge, one patient with MRSA presented two weeks post op and two patients with *S. epidermidis* presented two to four weeks post discharge.

The incidence of in hospital mortality was 4/34 (11.8%) for patients who developed DSWI compared with 102/5615 (1.8%) without DSWI (p = 0.003). There was no mortality in patients who underwent flap reconstruction. The mean duration of hospital stay was 25 days in the group of DSWI patients compared with 9 days in the group of patients without DSWI ( $p \le 0.01$ ).

A significantly greater proportion of patients in the DSWI group had diabetes which was controlled with oral medications compared with patients who did not develop DSWI (p = 0.021). The other risk factors for DSWI which were evident from the study included previous cardiac surgery (p = 0.038), BMI  $\geq 30$  (p = 0.041), LVEF  $\leq 30$  (p = 0.010), IABP (p = 0.028) and homologous blood usage (p < 0.001). The most prevalent risk predictor was homologous blood usage with 21/34 (61.8) DSWI patients requiring transfusion. Smoking status, use of preoperative steroids and type of cardiac operation did not affect the incidence of DSWI.

Table 3 outlines the treatment modalities of patients who developed DSWI. Four patients were omitted in the treatment approach due to incomplete records. All patients who received muscle flap reconstruction had prior failed debridement with or without rewiring. Furthermore all patients infected with S. aureus required muscle flap reconstruction. The most common treatment modality was bilateral pectoralis major muscle flap (BPMMF) with 11/30 (36.7%) requiring this form of flap reconstruction. A total of 4/16 (25%) patients who had flap reconstruction suffered a complication including donor site wound infection 1, haematoma – 1, acute recurrent deep wound infection – 1 and unstable sternum – 1. There was no partial or total flap loss. Mean follow-up of patients treated for DSWI was 3.2 years. During the follow-up period three patients passed away due to unrelated causes, six were unable to be contacted. Two patients had readmissions requiring minor wound management, none had persistent wound discharge. A further two patients complained of sternal pain on moderate activity but none had restrictions from activities of daily living. There was no effect on functional capability or shoulder movement on follow-up of patients who had received muscle flaps.

# Discussion

This is one of a few studies which have reported on rates of mediastinitis, risk predictors and management modalities from Australian institutions. A study conducted by Robinson et al. focused on mediastinitis rates in Victoria over

**Table 3.** Classification of DSWI Patients and Treatment Modalities.

Classification		Treatment Moo	dality		n = 30 (100)	
	Conservative	Surg. deb/rew.	PMAF	RAMF	BPMMF	CPMRAMF
I						
II						
IIIA	1 (3.3)	5 (16.7)				
IIIB		8 (26.7)				
IVA				3 (10)	11 (36.7)	1 (3.3)
IVB						
V			1 (3.3)			

Values are expressed as number of patients (percentage of DSWI patients who underwent treatment with complete records). BPMMF, bilateral pectoralis major muscle flap; CPMRAMF, combination pectoralis major and rectus abdominus muscle flap; PMAF, pectoralis major advancement flap; RAMF, rectus abdominus muscle flap; Surg. deb/rew, surgical debridement and rewiring.

an 11 year period recruiting close to 12,000 patients who underwent cardiac surgery in Victorian centres. This study reported an incidence of 1.3% for DSWI which is consistent with incidences reported by other centres throughout the world and particularly in Europe [1-4,7,13-17]. Furthermore our reported incidence of 0.6% compares favourable with other institutions and is similar to the lower end of the incidence spectrum of DSWI reported in the literature [1-4,7,13-17]. Our incidence has in fact remained steadfast over the past 10 years. Of greater importance and the objective of the study was to determine risk factors of DSWI in the cohort of patients that underwent cardiac surgery. We showed that diabetes, specifically controlled with oral medications; previous cardiac surgery and having a BMI  $\geq$  30 were important preoperative risk predictors for developing DSWI. Furthermore the necessity for homologous blood transfusion and IABP were important perioperative factors which contributed to the development of DSWI. Diabetes, being overweight and previous cardiac surgery are all well established risk factors as are the use of IABP and homologous blood requirements [1-3,6,7]. Of interest is that only diabetics managed with oral medication were at greater risk of developing DSWI, however those managed with insulin were not. The ability to achieve strict glycaemic control is more difficult in patients managed with oral medications and particularly perioperatively unless insulin infusion is utilised. Patients managed on insulin are more likely to achieve tighter control of their blood sugar levels and a study conducted by Furnary et al. conveyed this by showing that a continuous insulin infusion reduced the incidence of DSWI after cardiac procedures [18].

The impact of previous cardiac surgery is likely multifactorial. Longer perfusion times and in general operative times is an important risk predictor for DSWI [7,10]. Redo cardiac operations are associated with both the above risk predictors and at times greater need of blood transfusions. This consequently increases the risk of intraoperative wound contamination [21].

Patients' preoperative weight is an important modifiable risk factor identified and reported extensively in the literature. Lu et al. reported that a BMI  $\geq$  30 increased the risk of DSWI by more than double [19]. Overweight and obese patients pose a difficulty with primary sternal closure following cardiac surgery and have a higher risk of

wound dehiscence and sternal instability, thus increasing their risk of DSWI. This is a modifiable factor which needs further attention particularly in the elective setting where intervention can help to reduce its impact. BMI was not a predictor of DSWI in our study. We perform a figure of eight configuration for sternal closure, either three or four wires depending on the length of the sternum and at times heavier wires. We have not used double wires routinely however they are at our disposal.

Although previously reported and considered a modifiable risk factor, our study did not show smoking to be a risk predictor for developing DSWI. Patients were stratified based on status as either current or previous and this is important as studies have reported that current smoking can increase the incidence of DSWI by up three times. It is uncertain why a difference was not attained in this study.

The use of IABP is often a reflection of poor patient haemodynamics, increased monitoring lines associated with longer stay in intensive care units. Its use is a factor which may be difficult to prevent particularly in more complex procedures and unfortunately the burden of increased incidence of DSWI associated with its use is difficult to avoid. Limiting homologous blood usage however may be a factor which can be modified. Intraoperative surgical techniques aiming to limit blood loss and greater care during the operation are factors which could be addressed in attempting to limit the influence of this risk predictor.

The microbiological influence of DSWI in our centre was comparable to reported rates in the literature with *S*. aureus and S. epidermidis cultured most commonly from patients who developed DSWI [10]. It was interesting to note that all DSWI patients who cultured S. aureus required muscle flap closures for treatment. It is well understood that unlike coagulase negative staphylococci, S. aureus is not associated with sternal dehiscence or obesity [10]. DSWI due to S. aureus is due to operative infection and post operative infection may occur irrespective of the presence of sternal instability. DSWI due to coagulase negative staphylococci such as S. epidermidis presents more insidiously clinically and is associated with sternal instability more than any other bacterial aetiology. Deep sternal wound infection is thought to result from a small superficial infection of the sternal wound and subsequent spread inwards towards the mediastinal space due to sternal instability breaking the barrier between the presternal space and mediastinum [10]. Logically muscle flap closure would be expected more appropriate with coagulase negative bacteria due to sternal instability however there are currently no substantial studies on treatment modalities based on bacteriology or outcomes of various treatment modalities for DSWI stratified according to bacteriology. The use of muscle flap closure in patients who grew positive cultures for *S. aureus* is a reflection of the advanced classification of the DSWI for which many studies are surfacing rather than microbiology.

There is still significant debate and little consensus on the ideal treatment modality for DSWI. Treatment modalities vary from conservative therapy incorporating antibiotic therapy to surgical debridement and complete sternectomy with myocutaneous flap closure. We have over time become more aggressive in treatment and have involved the plastic surgeons earlier at the time of first debridement. At our institution there is no set of guidelines regarding the appropriate treatment modality for DSWI and surgeon preference to some degree dictates which approach is used. What is obvious however is that as the classification of DSWI according to El Oakley and Wright [12] becomes more severe (i.e. classifications II and above) then the use of myocutaneous flaps becomes more prevalent. This is mainly due to failure of the principal goal in these patients of sternal preservation. A study by Douville et al. reported that 6/32 (18.8%) of patients with El Oakley class IIB DSWI had failed attempts at sternal preservation [22]. Thus in individuals with more than one risk factor for DSWI, sicker and older patients, and those with S. aureus infection, a more aggressive treatment regime of sternal debridement and primary defect closure with myocutaneous flap will prove to be the mainstay of treatment. We have used Vac dressing only for superficial sternal wounds. Open infected sternums are dressed with normal saline as per plastics whilst awaiting flaps.

# Conclusion

DSWI is an uncommon but dreaded complication of cardiac surgery with significant morbidity, mortality and prolonged hospital stay. It is understood that our study was limited in that there is no long term follow-up of patients and the numbers of DSWI were small. However it is emphasised that preoperative strict control of blood sugar levels, encouragement of weight loss particularly in the elective setting and minimisation of blood product usage may reduce the incidence of DSWI. Finally it is hoped that with further centres publishing results on DSWI, a set of guidelines will be developed on the appropriateness of various treatment modalities.

# Conflict of interest

The authors declare that there is no conflict of interest in presenting this case.

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#### References

- [1] Elenbaas TWO, Hamad MAS, Schonberger JPAM, Martens EJ, Van Zundert AAJ, Van Straten AHM. Preoperative atrial fibrillation and elevated C-reactive protein levels as predictors of mediastinitis after coronary artery bypass grafting. Ann Thorac Surg 2010;89:704–9.
- [2] Ariyaratnam P, Bland M, Loubani M. Risk factors and mortality associated with deep sternal wound infections following coronary bypass surgery with or without concomitant procedures in a UK population: a basis for a new risk model? ICVTS 2010;August:18–21.
- [3] Steingrimsson S, Gottfredsson M, Kristinsson KG, Gudbjartsson T. Deep sternal wound infections following open heart surgery in Iceland. A population-based study. Scand Cardiovasc J 2008;42:208–13.
- [4] Sjogren J, Nilsson J, Gustafsson R, Malmsjo M, Ingemansson R. The impact of vacuum-assisted closure on long-term survival after post-sternotomy mediastinitis. Ann Thorac Surg 2005;80:1270–5.
- [5] Schimmer C, Sommer SP, Bensch M, Elert O, Leyh R. Management of poststernotomy mediastinitis: experience and results of different therapy modalities. J Thorac Cardiovasc Surg 2008;56:200–4.
- [6] Keib CN, Pelham JC. Mediastinitis following coronary artery bypass graft surgery: pathogenesis, clinical presentation, risks, and management. J Cardiovasc Nurs 2006;21(6):493–9.
- [7] Robinson PJ, Billah B, Leder K, Reid CM. Factors associated with deep sternal wound infection and haemorrhage following cardiac surgery in Victoria. ICVTS 2007;6:167–71.
- [8] Upton A, Smith P, Roberts S. Excess cost associated with Staphylococcus aureus poststernotomy mediastinitis. NZMA 2005;118(1210):1–3.
- [9] Sjogren J, Malmsjo M, Gustafsson R, Ingemansson R. Poststernotomy mediastinitis: a review of conventional surgical treatments, vacuum-assisted closure therapy and presentation of the Lund University Hospital mediastinitis algorithm. Eur J Cardiothorac Surg 2006;30:898–905.
- [10] Gardlund B, Bitkover CY, Vaage J. Postoperative mediastinitis in cardiac surgery – microbiology and pathogenesis. Eur J Cardiothorac Surg 2002;21:825–30.
- [11] Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Centres for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. Am J Infect Control 1999;27:97–132.
- [12] El Oakley R, Wright J. Postoperative mediastinitis: classification and management. Ann Thorac Surg 1984;38:415–23.
- [13] Baillot R, Cloutier D, Montalin L, Cote L, Lellouche F, Houde C, et al. Impact of deep sternal wound infection management with vacuum-assisted closure therapy followed by sternal osteosynthesis: a 15-year review of 23,499 sternotomies. Eur J Cardiothorac Surg 2010;37:880–7.
- [14] Immer F, Durrer M, Muhlemann KS, Erni D, Gahl B, Carrel TP. Deep sternal wound infection after cardiac surgery: modality of treatment and outcome. Ann Thorac Surg 2005;80:957–61.
- [15] Eyileten Z, Akar AR, Eryilmaz S, Sirlak M, Yazicioglu L, Durdu S, et al. Vacuum-assisted closure of bilateral pectoralis muscle flaps for different stages of mediastinitis after cardiac surgery. Surg Today 2009;39:947–54.
- [16] Satta J, Lahtinen J, Raisanen L, Salmela E, Juvonen T. Options for the management of poststernotomy mediastinitis. Scand Cardiovasc J 1998;32:29–32.
- [17] De Feo M, Gregorio R, Della Corte A, Marra C, Amarelli C, Renzulli A, et al. Deep sternal wound infection: the

- role of early debridement surgery. Eur J Cardiothorac Surg 2001;19:811–6.
- [18] Furnary AP, Zerr KJ, Grunkemeier GL, Starr A. Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. Ann Thorac Surg 1999;67:352–60.
- [19] Lu JCY, Grayson AD, Jha P, Srinivasan AK, Fabri BM. Risk factors for sternal wound infection and mid-term survival
- following coronary artery bypass surgery. Eur J Cardiothorac Surg 2003;23:943–9.
- [21] Milano CA, Kesler K, Archibald N, Sexton DJ, Jones RH. Mediastinitis after coronary artery bypass graft surgery. Circulation 1995;92:2245–51.
- [22] Douville EC, Asaph JW, Dworkin RJ. Sternal preservation: a better way to treat most sterna wound complications after cardiac surgery. Ann Thorac Surg 2004;78:1659–64.